

Society Reports.

PHILADELPHIA NEUROLOGICAL SOCIETY.

February 22d, 1897. President, Dr. Charles W. Burr, in the chair.

Dr. Alfred Stengel presented a case of "Arsenical Neuritis," occurring in a child who had been treated for chorea with Fowler's solution.

Dr. Francis X. Dercum.—The gait in this case is extremely interesting. It is like that of multiple neuritis in the adult. I would rather describe it as a "high-stepping gait" than as an ataxic gait. It also looks as though the extensors of the wrist were a little more flaccid than normal.

Another point which suggests itself is, whether or not all other causes than arsenic have been excluded. Is it possible that this has been a grip neuritis, or the result of a combination of several causes as, for instance, grip and arsenical poisoning? The rarity of the case makes one suspect some additional factor.

Dr. Charles K. Mills.—Is it necessary to regard this case as one of arsenical neuritis? Presuming that it is due to the influence of arsenic, may not the toxic effect have been exerted on the spinal centres rather than upon the nerves themselves? The absence of sensory symptoms, perhaps, points in this direction. We could have the train of symptoms presented here in a myelitic affection. This, however, would not alter the prognosis.

Dr. Alfred Stengel.—The condition of the arms was looked into at the time; but I could never demonstrate any loss of power in the extensor group, and I do not believe that there ever was any. I think that possible, coincident, casual factors, particularly influenza, may be excluded, as the patient was in the hospital when the condition developed, and it was in mid-summer, at a time when influenza was not prevalent.

Dr. Charles W. Burr read a paper, entitled: "A Case of Progressive, Neurotic, Muscular Atrophy." (To appear in a later number of this journal.)

Drs. F. X. Dercum and Isaac Leopold read a paper, en-

titled: "A Case of Primary Neurotic Atrophy" (see this journal, page 400.)

Dr. Francis X. Dercum.—I had the opportunity of seeing the case, which Dr. Burr reports, at the Orthopedic Hospital, independently of Dr. Burr, and came to the same conclusion, namely, that it was one of peroneal, muscular atrophy. I referred to this case at the November meeting of the Society. The case reported by Dr. Leopold and myself makes the fifth published in America. Sachs has reported two, Potts one, and Burr one.

Dr. Wm. G. Spiller.—Histologists were beginning to think they knew a little about the pathology of this disease. It has been supposed to be chiefly an affection of the peripheral nerves, although certain investigators have found lesions of the posterior columns and of the anterior horns of the cord. We were not prepared, however, for the interesting report which Oppenheim and Cassirer have recently published. You will notice in this reprint of their paper, that even the terminal nerve branches within the muscles are normal.

In 1893 Dejerine and Sottas published a paper, entitled: "Interstitial, Hypertrophic and Progressive Neuritis of Childhood," in which they presented the description of a disease not known at that time. It has many of the symptoms of primary neurotic atrophy, but in addition there are ataxia, fulgurant pains, nystagmus, myosis with Argyll-Robertson pupils, kyphoscoliosis, and great hypertrophy of the nerve trunks. I am able to present to you specimens of certain of the nerves from one of the cases published by these writers, and you will notice that there are scarcely any normal nerve fibres within the thickened bundles. Within the cord the ordinary degeneration of tabes may be found. Dejerine has recently published another case. There have only been four cases of this disease reported, and during my sojourn at the Salpêtrière I was able, through the kindness of Prof. Dejerine, to study the two patients now living, and the specimens from the case with autopsy previously published. I can assure you that it is a most interesting type of disease.

Dr. F. X. Dercum presented three cases of the family type of cerebral diplegia (see this journal, page 396).

Dr. Charles K. Mills.—These cases are similar to those published by me three or four years ago in one of the reports of the New Jersey Training School for Feeble-Minded Children. I referred to two or three groups of cases found there, in which other members of the family had similar trouble.

Dr. Wharton Sinkler.—Some time ago, I had a group of three cases in the same family, in which Dr. Osler took much interest. He regarded them as cases of Friedreich's ataxia. I was in doubt at the time, whether or not they should be

classed under that head, not only on account of the spastic condition which existed, but also because there were many symptoms of a distinctly cerebral origin. There was evidence of cerebral disease plus cord disease, although, apparently, the cord symptoms had been the earlier. There were four cases with the same symptoms in the family, but I saw only three of them.

Dr. John K. Mitchell.—I have notes of two cases of a paraplegic type without mental affection. Both occurred at an early age, and both followed slight attacks of acute disease. The disease began in the second year, and both have since had advanced spastic paraplegia. They are now sixteen and eighteen years of age. There were eleven children in the family, all of the others being in good health, with the exception of one who has epileptic attacks.

Dr. J. P. C. Griffith.—I would like to ask Dr. Dercum, whether he would not distinguish between heredity, or family, spastic paraplegia and Little's disease? He speaks of Van Gehuchten as discussing the latter, but, as I understand the articles, the recent writings of this author discuss not *hereditary* spastic paralysis, but *congenital* spastic rigidity or Little's disease. Sachs rightly, I think, claims that the two are to be sharply differentiated. Van Gehuchten was not considering at all such cases as Dr. Dercum reports. He goes even farther and limits Little's disease to the cases in which not merely an accident during birth, but a developmental defect, the result of premature birth, is also operative. Hereditary, spastic paraplegia very commonly first shows itself later in life. It is not congenital, as Little's disease is.

Dr. Dercum has not mentioned the interesting cases of the family form of spastic paraplegia reported by Neumark in 1893. I notice he has recently made a further contribution to the subject.

I have now under observation two children which strongly suggest the presence of hereditary spastic paralysis, but I have not yet studied them sufficiently to be able to publish them as such.

Dr. John K. Mitchell.—To say that this is or is not a congenital condition is merely a question of terms. The nervous systems of these children are, as it were, hung on hair triggers, and a very slight cause is capable of exciting great disturbance. After the first symptoms are seen, this disease develops too rapidly to be a degenerative disease without previous disorder. Some primary deficiency must necessarily be at the bottom of a trouble which appears so rapidly and starts from such trifling causes.

Dr. M. V. Ball.—I wish to report two cases. The older child, aged seven years, is a congenital idiot with a spastic con-

dition. The younger child is feeble mentally, and we may look for a spastic condition in its case. It does not yet walk. I believe that it is not uncommon to find several congenital idiots in the same family with a spastic condition in some of them.

Dr. F. X. Dercum.—Cerebral diplegia arises from two very different causes, first, cerebral diplegia may be due to imperfect or feeble development of the neurons of the motor area. Here spontaneous degeneration may lead to the appearance of spastic palsy, or the vitality of the neurons may be so low that they perish from very slight causes, for example, the infection of measles. Secondly, there is diplegia due to trauma received during birth (dystocia). To this group the term "Little's disease" should be restricted. It may occur in children whose nervous system presents a normal degree of resistance.

Dr. David Riesman presented two cases of brachial tremor. The first patient, a married woman, aged 54, has had a fixed torticollis for 10 years, which she attributes to sleeping one night in a draught after having washed her hair. Four years ago she suffered from an attack of rheumatism, and has been more or less rheumatic from that time. Since January, 1896, she has had a rapid rhythmic tremor of the right forearm, ceasing during voluntary movements and during sleep. The movements are 6 to the second. There are no tremors in other parts of the body; the knee jerks are exaggerated; ankle clonus is not present, and sensation is normal.

The patient states that three months before the onset of the affection she saw a man in a car who was suffering from marked tremor; she was greatly startled, and the sight made a strong impression on her mind. Before the onset of her trouble she was also subjected to great mental strain by her husband's intemperate life.

The speaker considered the case probably one of hysteric tremor.

The second case was a married woman, 40 years old, who for 14 years had had a violent spasm of the right arm. It came on after her second confinement, during the lying-in. She was frightened by a fire in the neighborhood, and for five days had generalized spasms of all four extremities. These movements subsided in all the limbs except the right arm. For the last six years the movements have stopped scarcely more than 15 minutes at a time, when the patient was in repose, though they always cease during work. Excitement greatly intensifies them. She suffers also from palpitation, and on examination was found to have a double aortic murmur.

The movements of the arm are of large amplitude, and when the hand, during the spasm, is resting on any surface, a loud, patting noise is produced.

On account of fugitive rheumatic pains, the patient was at

first placed on a mixture of potassium iodid and sodium salicylate. She improved greatly, and on her third visit the tremor had ceased, and since then, September 15th, 1896, she considers herself cured, as far as the tremor is concerned. Considering that the affection had lasted 14 years, and had been constant for the past 6 years, it can only have been hysteric in nature, the recovery being attributable to the influence of suggestion.

Dr. F. X. Dercum.—The first case would appear to be one of paralysis agitans, though somewhat atypical. There is rigidity of the back of the neck, slowness of movement and fine tremor, arrested by volition. The tremor is not typical in distribution, but this, as is well known, varies greatly. Festination is not always present in paralysis agitans.

Dr. Wharton Sinkler.—I agree with Dr. Dercum that this is probably a case of paralysis agitans. I had a case in an elderly man who had been subjected to a severe shock by seeing a falling elevator shoot past him as he stood by an opening. Tremor began at once in the right arm and never ceased afterward except under voluntary effort. Many other instances are on record of paralysis agitans taking its origin in sudden mental impressions, such as fright, grief, or injury.

Dr. D. Riesman.—I do not think that the condition of the neck can be taken into account as sustaining a diagnosis of paralysis agitans. The neck has been this way for ten years. There has been no complaint of sweats or vasomotor symptoms. While I cannot bring any other arguments against the diagnosis of paralysis agitans, yet time will settle the question.

Dr. Wm. G. Spiller presented an unusual case of hemiplegia (see this journal, page 391).

Dr. Charles K. Mills.—It is not probable that this case is one of hysteria. I believe that the man is suffering from a hemiplegia of organic origin, although he may have hysterical symptoms, but these are epiphenomena. He may have had a hemorrhage, not very large, into a certain part of the internal capsule. The paralysis of his arm and leg is to my mind that so often seen in organic hemiplegia; so also is the tremor, which is like that which has been described as hemiplegic or post-hemiplegic tremor. It will be noticed that he has some facial hemiparesis, although this is not very marked. The contractions in arm and leg do not seem to be of the hysterical type.

Most conclusive of all to me is the foot clonus which is shown by this patient; it is the clonus of organic hemiplegia. Of this I feel certain from many years' experience in the examination of hysterical and organic cases, and I would like to strongly emphasize the importance of the symptom ankle clonus in the differentiation of hysteria from organic disease. While the knee jerk may be, and frequently is, excessive in

cases of grave hysteria of the hemiplegic or of other types, I have never yet seen a case in which the persistent clonus, which is so significant of certain forms of organic disease both of the brain and spinal cord, was present. Occasionally what might be termed a *pseudoclonus* is observed. Gowers has described this as a spurious clonus, and believes that it is due to a half voluntary contraction of the calf muscles.

In the vast majority of cases of hysterio-traumatism, of which I have examined a large number, and especially during the last four years, even this form of spurious clonus is usually absent. It is misleading for writers on hysteria to speak of the presence of ankle clonus in such a way as to give the impression that it may be of a well-marked and persistent character like that which is seen in organic disease. Several important matters must be borne in mind in this connection, as, for instance, the fact that organic disease, either spinal or cerebral, is often associated with hysteria or hysteria with organic disease, and the presence of a true clonus in such cases may have misled the diagnostician. I have seen a few cases of this kind: one, for instance, in which the persistent ankle clonus was present, and in which the diagnosis of hysteria was made and adhered to for some time, but the case eventually proved to be one of organic spinal disease, probably lateral sclerosis. In another case, which fell under my observation, and which has been cited by one of my colleagues as proving that ankle clonus is sometimes present in grave hysteria, it was believed that the patient had angular curvature with compression of the cord. It was supposed that she could not walk, and for a long time she had been in a wheeling chair. On one occasion, after the administration of some remedy, practically a placebo, she got up and walked. This case was, I believe, one of partially cured caries and local pachymeningitis. Although the patient had partially recovered, the cord at a certain level was probably still subjected to some irritation and possibly compression, the latter not being sufficient to destroy its transmitting functions. This case may have been one of hysteria, but the hysteria was probably imposed upon an organic disease. Ankle clonus of the genuine persistent type was present, but I do not believe that it proves anything with regard to the significance of the symptom in the diagnosis of hysteria.

Dr. James Hendrie Lloyd.—Some time ago I was called in consultation in this case. The rather unusual question had arisen, whether it was desirable, on account of the extreme tremor, to amputate the arm. Amputation of the arm for post-hemiplegic tremor was not a procedure which I could endorse, and the project was finally abandoned. I demonstrated that, the trouble being of central origin, amputation of the arm would remove only part of the structures that were affected,

and was entirely unjustifiable. The demand for amputation, it seems, had been made by the patient himself, who declared that he would commit suicide if something were not done for his relief.

When I examined the man a year ago, I had no hesitation in making a diagnosis of organic lesion of the brain, and I said then that this tremor was only a grave type of posthemiplegic tremor. I still think that that is the correct diagnosis. I am ready to believe, however, that there may be some hysterical stigmata complicating the case, as is not unheard of.

I would call attention to the fact that the history of hemianesthesia, paralysis of all four extremities, and amnesia for languages, which were said to be present in the early stages of the case, is to a large extent based upon hearsay evidence. We have no very authentic data as to the man's condition at the commencement of the attack. I wish to go on record as having expressed the opinion a year and a half ago, that the man had organic hemiplegia. I base this diagnosis upon the type of the hemiplegia, which is organic rather than hysterical; *i. e.*, it is worse in the arm than in the leg, and is accompanied with characteristic and exaggerated reflexes (especially a well-marked ankle clonus); that the tremor is unilateral and confined to the affected side; that there is not a true hysterical hemianesthesia with involvement of mucous surfaces; and, finally, that there is no contraction of the visual fields satisfactorily demonstrated. If this man has not a hemiplegia of organic origin, then I do not see how henceforth we are to distinguish organic hemiplegia.

Dr. F. X. Dercum.—I regard this case as unequivocally one of traumatic hysteria. The fact that he had hemianesthesia is very suggestive; also the time that elapsed before the onset of the symptoms. It is not probable that he had a hemorrhage so long after the accident. I have seen ankle clonus in traumatic hysteria, and have records of such cases. In my mind the presence of an ankle clonus militates in no wise against the diagnosis of hysteria.

Dr. Wharton Sinkler.—The case seems to me one of traumatic hysteria for the following reasons: The man was totally paralyzed; he was in a condition of inhibition of voluntary function, so that, although he could hear sounds, he was unable to raise himself from the floor or to call for help. The aphonia is also typical of hysteria and is quite unlike organic brain disease. The history does not belong to that of any form of hemiplegia from organic disease. I am certain that ankle clonus may exist in hysteria, particularly in traumatic hysteria.

Dr. A. Ferree Witmer.—The involvement of the fibres of the seventh nerve is of extreme interest. I should like to mention a similar condition that I saw in a young woman in the

eighth month of pregnancy, in whom there was evident facial palsy. There had been a marked element of hysteria in the family for three generations. I regard this as a case of hysterical facial palsy, due to the excitement attending her condition.

Dr. F. Savary Pearce.—In regard to the diagnosis of organic disease in this man, I can cite a case of severe muscular strain producing hemorrhage from the lateral sinus, which was seen at the autopsy. This man presented to-night was also subjected to great muscular effort. In Dr. S. Weir Mitchell's practice I have recently seen a woman of 56 years, who in March, 1896, had a hemorrhage involving the speech centre, from which she slowly recovered. She was conscious during the onset of the attack and could speak. The patient had right hemiplegia. I mention this case as an evidence that there may be organic paralysis without mental disturbance.

Dr. Wm. G. Spiller.—I am sorry that no one has spoken of the peculiar disturbance of speech. The patient lost his power of speaking in all languages except one. I can recall a similar case which I saw in the clinic of Krafft-Ebing in Vienna. The diagnosis between hysteria and organic disease in that case was not easy. Such a partial loss of languages may, of course, be organic.

I do not think we should regard the predominance of the contracture in the upper extremity necessarily as due to organic disease. It must be borne in mind that the man is, and has been, very excitable; and that he attempted to take his life a short time ago. A few years ago he had a tremor in the right lower limb very similar to that now seen in the upper. This has entirely disappeared, except when the patient is excited. The tremor in the upper limb is, to my eye, characteristic of hysteria, so much so that the diagnosis of hysteria presented itself to my mind the instant I saw the man. True ankle clonus is uncommon in hysteria, but Gowers does not deny its existence. He says, it is "extremely rare." I have presented this case as an unusual form of hemiplegia. Shall we make ankle clonus a test for organic disease? I would hesitate to call every case organic in which it might be found. The severe mental strain, the late appearance and mode of development of the hemiplegia are in favor of the diagnosis of hysteria. The man is very intelligent, and I see no reason for doubting the account which he gives of his disease. His statements in no way conflict. We must remember that certain stigmata of hysteria may disappear after a number of years. The slight disturbance of consciousness during the first hours of the paralysis, the ability to notice everything about him, even to recall the hour, the aphonia, the complete paralysis of all four limbs form a combination of symptoms more like that of hysteria than of organic disease. Shall we disregard the clinical

history in a given case, simply because it does not agree with our views? On this the diagnosis in the present case must chiefly rest.

Dr. James Hendrie Lloyd reported a case of cerebellar tumor and presented the specimen.

Dr. H. A. Hare read a paper, entitled: "A Report of a Case of unusual Edema in Hemiplegia. (To appear in a later number of this journal.)

Dr. James H. Lloyd.—I would like to ask Dr. Hare if he took into account the possibility of this being a hysterical edema. We sometimes have remarkable examples of edema in hysteria. I saw a marked instance of this in a medico-legal case of brachial monoplegia due to an electric shock. The arm was flaccid and presented an extraordinary blue edema, such as has been described by Charcot.

Dr. F. Savary Pearce.—May not the mitral disease account in part for the edema in the paralyzed arm where all function is sluggish? Dr. Hare tells me that the patient at one time had edema of other parts.

Dr. H. A. Hare.—This was not a blue edema.

ANALGESIA OF THE ULNAR NERVE IN THE INSANE.

Dr. Arrigo Giannone gives a detailed account of his investigation regarding the condition of the ulnar nerve, in July No. of *Rivista di Patologia Nervosa e Nuntale*; this work was investigated by the researches of Biernachi, who found analgesia of this nerve in locomotor ataxia.

In 47 cases of *Dementia Paralytica*, there was analgesia of both nerves in 21 cases (males); analgesia of one side in 2 cases (males); there was lessened sensibility in 10 cases (males); normal in 6 cases (males); analgesia in 2 cases (females); lessened sensibility in 2 cases (females); normal in 4 cases (females); recapitulating, the ulnar symptom was found in 53.2 per cent, lessened sensibility in 25.6 per cent., and the nerve was normal in 21.2 per cent.

In an examination of 210 insane, the ulnar symptom was found in 53, or 24.9 per cent.; lessened sensibility in 53, or 24.9 per cent.; normal in 107, or 50.9 per cent. Similar researches were made in regard to patients with locomotor ataxia with the following results: Out of 15 patients the ulnar symptom was present in 9, or 60 per cent.; lessened sensibility in 2, or 13.3 per cent; normal in 4, or 26.7 per cent.

In regard to its importance in locomotor ataxia the author concludes:

1. That the analgesia of the ulnar nerve is not to be considered in the same category as the other symptoms of locomotor ataxia.

2. That considered by itself, it is of little importance, being met with much less frequency than the Argyll-Robertson pupil or Westphal's symptom.

3. That it is not pathognomonic of locomotor ataxia, but occurs in other diseases of the brain and nervous system. KRAUSS.